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SHORTER ARTICLES AND DISCUSSION

INHERITANCE OF CONGENITAL CATARACT

CATARACT is the opacity of the eye caused by a faulty formation of the lens. Certain forms of cataract are congenital and hereditary. Other forms which appear later in life may either be hereditary or due to pathological causes.

In the normal eye the delicate fibers which go to make up the lens are glued together along their sides and at their ends where they unite in lines radiating from the poles of the lens to form a completely transparent body. Anything which prevents the perfect conjunction of these fibers causes a defect in the transparency of the lens. This imperfection has been compared by Harman (2) to the white spots in the finger nail, caused by slight injuries to the nail bed, and he has shown it to be correlated with faulty formation of the dental enamel.

There are various causes for the inhibition of proper lens development, and these give rise to different forms of cataract. Only those forms have been considered here which are congenital.

The most common form of congenital cataract is the lamellar, perinuclear or zonular cataract. This manifests itself as a dark circular disk with the density increasing from the center to the perimeter, forming characteristic zones. These zones are flecked by small wedge-shaped dashes arranged regularly in a spoke-fashion about the disk. The disk is located between the nucleus of the lens and the cortex; and is caused by a thickening of the layers at that place.

Discoid cataract is a slight form of the lamellar, less than 4 mm. in diameter, and located at the posterior pole of the lens. The opacity is uniform throughout, but is not easily visible. (It is sometimes confused with anterior polar cataract, of which the origin is not definitely known, but which is not congenital.)

Coralliform or axial cataract, *cataracta fusiformis*, is an opaque line running through the lens from anterior to posterior pole with a spindle-shaped swelling towards the center of the lens.

Anterior and posterior cortical cataract, *cataracta corticallis,* where the opacity takes a more or less geometrical outline, *cataracta punctata,* formed by minute white dots scattered uniformly through the lens or grouped in the anterior cortical layers, and other forms of circumscribed, stationary, lenticular opacities which though rare are known to be congenital (3), have also been used in the compilations given here.

Senile cataract, which also seems to be hereditary, and those forms of cataract arising from lesions, diseases of the eyeball, and certain general diseases such as cholera and tetany, have been omitted.

Although congenital and other forms of cataract have long been considered by the medical profession to be influenced by heredity, no definite analysis was made until 1905, when the first paper by Nettleship (1) appeared. Nettleship's data have been the basis of Bateson's (9) conclusion that the abnormality is inherited as a dominant character. Bateson acknowledges that normal parents have produced abnormal children, but these cases he explains as either *origin de novo,* or due to faulty classification of the parents, who in reality may have been slightly affected with cataract.

Davenport (3) has followed Bateson's conclusion in regard to the inheritance of cataract, and makes the eugenic recommendation that unaffected parents from affected stock may marry without fear of producing abnormal children.

In the "Treasury of Human Inheritance" Harman (2) gives one hundred genealogical tables dealing with congenital cataract. Each table represents two or more generations with a detailed account of the condition of each individual in regard to congenital defects of the eye. The data used in this paper have been taken from these tables. Only those families are used in which there is no doubt as to the condition of the parents or the children in respect to the abnormality, and where there is no question as to the total number of children in each family. After discarding all the doubtful cases, and picking a sibship with its parents from the table as a family, there is left a total of one hundred and twenty-five families which are classified into three different categories, as follows: (*A*) both parents normal with at least one abnormal child; (*B*) one parent normal, the other affected with some form of congenital cataract, with at least one abnormal child; (*C*) both parents abnormal, giving only abnormal children.

There are 31 families with both parents normal which give some abnormal children. In a total of 153 children from these families, 61 are affected with cataract. This suggests that the character is more likely to be inherited as a recessive than as a dominant. Surely it is not possible to explain so many cases as *origin de novo* or as due to faulty classification of the parents.

In the second category given above (B), where one of the parents is affected and the other normal, the number of defective children would be expected to be approximately the same whether the character was inherited as a dominant or a recessive.

If the abnormality is considered as a recessive character, the ratio of 61 affected to 92 unaffected, already spoken of as having been obtained in the first category of families, shows an excess of recessives over the simple Mendelian expectancy for a monohybrid. This is to be expected since the criterion for including any family in the tabulation is the production of at least one abnormal child. In families with a small number of children it is probable that in some cases only normal children are produced in matings of heterozygote by heterozygote which should give, on the average, one fourth recessive. The observed results must then be compared to a modified Mendelian ratio which will allow for the omission of all-normal progenies. Such ratios have been calculated by Apert (4) and by Wright (5). The expected proportions given here are calculated according to the method given by the latter.

The proportion of recessives varies according to the number of children in the family and ranges, for a three-to-one ratio, from 100 per cent. in families with one child to very nearly 25 per cent. in families with fifteen children. The proportion is calculated from the formula

$$X = \frac{1}{4[1 - (\frac{3}{4})^N]},$$

where N is the number of children. Since the criterion for including any family is the production of one abnormal child, all families with one child must have 100 per cent. abnormal children. The proportion decreases, according to the law of chance, as the number of children in the family increases, finally reaching 25 per cent. as the number of children becomes large.

Table I compares the results obtained with the theoretical expectancy, worked out according to this method.

The method used for testing the agreement of the observed result with the theoretical is the one given by Pearson (6) and Elderton (7). It was originally used to test various series of bio-

TABLE I

NORMAL × NORMAL (BOTH HETEROZYGOUS — NN × NN)

Size of Family N	Number of Families	Total No. of Children	Calculated Proportion Recessive X	Calculated Number of Recessives C	Observed Number of Recessives O	$O - C$	$\frac{(O - C)^2}{C}$
1	2	2	1.0000	2.00	2	.00	.000
2	4	8	.5714	4.57	6	1.43	.447
3	6	18	.4324	7.78	13	5.22	3.502
4	5	20	.3657	7.31	8	.69	.065
5	0	0	.3278	.00	0	.00	.000
6	7	42	.3041	12.77	19	6.23	3.039
7	1	7	.2885	2.02	2	− .02	.000+
8	2	16	.2778	4.44	5	.56	.070
9	1	9	.2703	2.43	1	−1.43	.841
10	2	20	.2649	5.30	3	−2.30	.998
11	1	11	.2610	2.87	2	− .87	.263
	31	153		51.49	61		9.225

$$N = 10.$$
$$P = .418$$

	Normal	Abnormal	Per Cent. Abnormal
Observed	92	61	40
Calculated	102	51	33
Difference	10	10	7

logical measurements. Attention has been called to the application of this method of testing theoretical Mendelian ratios by Harris (8).

By calculation from the data given in Table I the measure of agreement, or "*P*," is .418. "*P*" is a value ranging from 0 to 1, proportional to the closeness with which the observed facts agree with the theoretical. In this case in four times out of ten, random samplings of similar data would give results deviating more widely from the theoretical. A possible explanation for this rather wide discrepancy will be given later.

The children from matings of normal by abnormal are given in Table II.

A total of 448 children from 90 families is used. 232 children out of the 448 are found to be defective, whereas 238 are expected according to the modified Mendelian ratio.

As before, the criterion for including any family in the tabulation is the production of at least one affected child. Only in this way can the matings of heterozygous dominants, *Nn* (normal), with homozygous recessives, *nn* (abnormal), be distin-

TABLE II

NORMAL × ABNORMAL (NN × NN).

Size of Family N	Number of Families	Total No. of Children	Calculated Proportion Recessive X	Calculated Number of Recessives C	Observed Number of Recessives O	$O-C$	$\frac{(O-C)^2}{C}$
1	9	9	1.0000	9.00	9	.00	.000
2	11	22	.6667	14.67	20	5.33	1.936
3	10	30	.5714	17.14	19	1.86	.201
4	12	48	.5333	25.60	24	−1.60	.100
5	10	50	.5161	25.81	27	1.19	.054
6	8	48	.5080	24.38	23	−1.38	.078
7	11	77	.5039	38.80	40	1.20	.037
8	9	72	.5019	36.14	31	−5.14	.731
9	3	27	.5009	13.52	15	1.48	.162
10	3	30	.5005	15.02	9	−6.02	2.412
11	2	22	.5002	11.00	10	−1.00	.090
12	0	0	.5001	.00	0	.00	.000
13	1	13	.5001	6.50	5	−1.50	.346
	90	448		237.58	232		6.147

$N = 12.$
$P = .862$

	Normal	Abnormal	Per Cent. Abnormal
Observed	216	232	52
Calculated	210	238	53
Difference	6	6	1

guished from the matings of homozygous dominants, *NN*, with recessives. In the former matings approximately 50 per cent. of the children are expected to be abnormal. In the second case only normal children are expected, who should all be heterozygous for the abnormality. It is entirely possible in small families that matings which should give part abnormal offspring might give only normal children; but all these matings are excluded, for no distinction can be made between them and the more usual matings of abnormal with homozygous normal, also giving only normal children.

Having thus excluded part of the data, the modified ratio is calculated as before, except that in this case it applies to a one-to-one ratio, instead of a one-to-three. It ranges from 100 per cent. in families with one child, to very nearly 50 per cent. in families with fifteen children, and is calculated from the formula

$$X = \frac{1}{2[1 - (\frac{1}{2})^N]}.$$

The agreement of observation with expectancy is very close.

"*P*" having a value .862 means that, in nearly nine cases out of ten, other random samplings would deviate more widely from the theoretical.

The critical test as to whether or not congenital cataract can be considered as a simple recessive character lies in the matings of abnormal by abnormal. Families of this kind should have only abnormal children. Only three such matings are available. In two of these, five and two children, respectively, are the total numbers produced; and these are all abnormal. The other case is a doubtful one. Both parents are classified as having discoid cataract; one is given as seriously affected, the other only slightly so. Seven children are given for this mating: two are abnormal, and the others apparently normal. Three of these five died in infancy, so that their classification is doubtful, but there is no question as to the others. Assuming, as Bateson does in his cases, that there has been a faulty classification of the parents, that the parent given as slightly affected is not congenitally defective, but adventitiously so early in life; then this one discrepancy might be conveniently overlooked.

Another explanation may be found, however, in the fact that heterozygous individuals sometimes show the recessive character. Cases are known where a small percentage of heterozygous individuals show the recessive character, although the homozygous and heterozygous dominants are generally indistinguishable. If such is the case here, the slightly affected parent is heterozygous, and the occurrence of normal children is expected.

This assumption also helps to explain the deviation of observed results above the theoretical in Table I, and their deviation below the theoretical in Table II. If heterozygous individuals are sometimes classified as recessives, it would affect the classification of both the parents and the children. A few matings of abnormal (heterozygote showing the recessive character) by normal would be included in the category *B* which rightfully belong in category *A*. Thus matings giving a one-to-three ratio would be included among matings giving a one-to-one ratio. This would tend to reduce the observed results below the expected. A faulty classification of the children would tend to raise the results, but this would not be as strong a deviating factor as when influencing the parents and therefore a number of children. Thus the balance would tend slightly toward a decrease in the regular expectancy; a result which fits well with that obtained in Table II.

In the matings of normal by normal, there is, of course, no opportunity for this error to influence the classification of the parents, since abnormals, whether heterozygous or not, would not come here. But with the children, the number with the recessive character would be raised above the regular expectancy; a result which coincides with that in Table I.

If with more matings of abnormal by abnormal it is found that, with a few exceptions, only abnormal children are given, the evidence that cataract is a recessive character rather than a dominant will be fairly conclusive. It seems rather strange that congenital cataract manifesting itself, as it does, in such different ways, should be determined by a single unit factor. These things, however, must be explained in the simplest possible manner; an attempt to work it out with two or more factors would introduce great complications, and be practically impossible with the data as they have been gathered heretofore. The fact that a recessive character may not be recognized, for it occurs in mass data in a greater proportion than would be expected at first, should be noticed. Finally the approximation of the results obtained with those expected from the single unit factor form the best reason for its acceptance.

That certain geneticists should have laid down eugenic rules based on the inheritance of this character as a dominant is, at the very least, unfortunate. It is not only because of a mistake in the method of inheritance, but such rules should never be made until the exact hereditary processes are positively known, since such practises are likely, not only to bring discredit upon the science, but to injure people who endeavor to follow them in the regulations of their lives.

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HUXLEY AS A MUTATIONIST

ELSEWHERE I have pointed out that Galton[1] held with equal firmness to continuity and discontinuity in variation, and that the American horticulturist and botanist, Thomas Meehan,[2] held clear mutationist conceptions which he supported by accurate observations of variations in many plants. It seems worth while to add a note on the attitude of Huxley with regard to this question.

Whenever Huxley expressed himself on this matter he usually took occasion to say explicitly that he could see no reason why variations should not be discontinuous as well as continuous, and one of the few points on which he differed from Darwin was in ascribing greater significance to such marked changes. Several statements of his position in this matter are found in his volume of essays entitled *Darwiniana.*

Thus he says (p. 77):

> Mr. Darwin's position might, we think, have been even stronger than it is if he had not embarrassed himself with the aphorism "*natura non facit saltum,*" which turns up so often in his pages. We believe, as we have said above, that Nature does make jumps now and then, and a recognition of the fact is of no small importance in disposing of many minor objections to the doctrine of transmutation.

Elsewhere (pp. 34, 404) Huxley refers to the well-known Ancon sheep, which originated from a single ram in the flock of a Massachusetts farmer named Seth Wight. The story of this breed of sheep is told in a letter from Col. David Humphreys to Sir Joseph Banks, then President of the Royal Society.[3] The farmer kept a flock of 15 ewes and one ram on the banks of the Charles River, at Dover, Mass., 16 miles from Boston. In 1791 a ram

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[1] "Galton and Discontinuity in Variation," AMER. NAT., *48:* 697–699, 1914.

[2] "An Anticipatory Mutationist," AMER. NAT., *49:* 645–648, 1915.

[3] Humphreys, D., 1813, "On a New Variety in the Breeds of Sheep," *Phil. Trans. Roy. Soc.,* 1813: 88–95.